TROPICAL (FIBRO-CALCULOUS) PANCREATIC DIABETES

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In tropical countries special forms of diabetes associated with protein calorie malnutrition have been reported. The recent WHO study group report has set apart a category of diabetes that is distinct from insulin dependent diabetes mellitus (IDDM) and non-insulin dependent diabetes mellitus (NIDDM), the two major forms of diabetes seen in developed countries of the western world. This type of diabetes has been designated as malnutrition related diabetes or MRDM. Under the category of MRDM two separate entities have been included. These are fibrocalculus pancreatic diabetes (FCPD) and protein deficient pancreatic diabetes (PDPD). There are problems with the exact definition of PDPD. For this reason most of the studies done by us have been on FCPD. In this article some of the recent studies in this field will be summarised.

Historical Landmarks in the Description of FCPD

The syndrome recognised today as FCPD was first described by Zuidema in Indonesia. Zuidema's cases were malnourished and had evidence of pancreatic calcification on abdominal X-ray. A subsequent report from Shaper described the occurrence of this entity in Uganda. Reports of FCPD have also been published from Zaire, Nigeria, Ghana, and other countries from Africa. From Asia, FCPD has been reported from Thailand, Bangla Desh, Sri Lanka and the largest reports of this has come from southern India. Geevergese first pointed out the common occurrence of this disease in Kerala and to date has over 1700 patients in his series. FCPD has also been reported from the neighbouring states such as Tamil Nadu, Karnataka and Andhra Pradesh. Cases have also been described from Orissa, New Delhi, Pune and Bombay.

Criteria for Diagnosis of FCPD

Based on the above studies, Mohan et al have proposed the following working definition for FCPD:
1) Diabetes must be present according to the criteria of the National Diabetes Data Group (NDDG) or WHO study group report.
2) The disease must be present in a tropical country.
3) There must be evidence of chronic pancreatitis. For this the single most important diagnostic criterion is presence of pancreatic calculi in the plain X-ray of the abdomen. In cases where calculi are absent at least 3 of the following must be present:
   a) Presence of structural pathology in the pancreas such as fibrosis, ductal dilatation etc. as demonstrated by CT scan, ultrasound or ERCP;
   b) A definite history of abdominal pain from childhood
   c) Steatorrhoea or
   d) Abnormal exocrine pancreatic function tests
4) Other causes of chronic pancreatitis such as

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alcoholism must be excluded.

**Prevalence of FCPD**

There are no studies as yet which have described the prevalence of FCPD in the whole population. The reason for this is that for the diagnosis of FCPD it is necessary to demonstrate the presence of pancreatic calculi on a plain X-ray of the abdomen and the problems of doing large scale abdominal X-rays in the field setting in a country can well be imagined. Surprisingly there is paucity of data even from diabetic clinics on the prevalence of FCPD with respect to the total number of diabetic patients. At the Diabetes Research Centre, Madras, 97% of all patients have NIDDM form of diabetes and FCPD constitutes less than 1% of all diabetic patients. If only young diabetics i.e. those with age at diagnosis below 30 years of age are considered, about 4% of patients would be classified as FCPD.

**Heterogeneity in Clinical Profile of FCPD**

Earlier studies on FCPD have suggested the following characteristics of FCPD:
1) Patients belong to the lowest socio-economic strata of society
2) There is evidence of gross emaciation and protein calorie malnutrition
3) The diabetes is severe and insulin requiring
4) In spite of large insulin requirements patients are not prone to ketosis and
5) Cassava (tapioca) ingestion is present and has been suggested as an aetiological factor in the pathogenesis of the disease.

A recent study from the Diabetes Research Centre, Madras has brought to light the heterogeneity in the clinical presentation of FCPD. We found that while almost all patients were lean, overt malnutrition was seen only in 25% of patients. While the majority of patients required insulin for control of diabetes, some responded to oral agents. Again, while the majority of the insulin treated patients were ketosis resistant, a sub-group were prone to ketosis. Finally, cassava ingestion was uncommon in the cases seen at Madras. These factors point to heterogeneity within the FCPD syndrome.

**Phenomenon of Ketosis Resistance in FCPD**

Several theories have been put forward to explain the infrequency of ketosis in FCPD. These include lower adipose tissue mass, low fat intake in the diet and low plasma glucagon levels. A recent study from our centre has offered another explanation. It was seen that the patients with FCPD had plasma C-peptide levels that were intermediate between that seen in classical IDDM patients and those with NIDDM. Thus while they lacked sufficient beta cell reserve to respond to oral agents they had enough endogenous insulin to protect them from developing ketosis in the basal state. There was also a fairly good correlation between the response to treatment and C-peptide levels.

**Vascular Complications in FCPD**

Earlier studies had suggested that vascular complications were rare in FCPD. We have shown that microvascular complications are as common in FCPD as in the primary forms of diabetes. Both proliferative retinopathy and maculopathy, the two sight threatening forms of retinopathy are seen in FCPD. Nerve conduction studies showed that the occurrence of neuropathy was also frequent as were nephropathy and renal insufficiency. Left ventricular dysfunction also occurred in FCPD. Macrovascular complications on the other hand were less common. This may be related to the lower body mass indices, the relative youth of the patients and the low lipid levels.

**Ultrasonographic Studies in FCPD**

Real time B mode ultrasonography was performed in patients with FCPD and mat-
ched groups of patients with IDDM and NIDDM and healthy control subjects. It was seen that the size of the pancreas was reduced in FCPD patients. The presence of calculi and fibrosis could be picked up by this non-invasive technique. Additional features made out by ultrasonography include ductal dilatation and intraductal calculi in some cases. In a few cases, where the calculi were not detected on X-ray, the presence of fibrosis on ultrasonography helped to spot cases with FCPD.

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It seems to me that people in the Buddha's time were more advanced in tolerance and compassion than we are, although they were not so advanced in technology and science.

— Pandit Jawaharlal Nehru